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Dysregulation of Intracellular Ca^{2+} and Camp Signalling: Plausible Targets for Neurological Disorders

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Editorial

My current field of research involves the study of the interaction between Ca^{2+} and cAMP signalling pathways, including its role in neurological disorders. The scientific literature now clearly accepts this interaction as a fundamental cellular process, which is also involved in synaptic transmission mainly by controlling neurotransmitter release [1]. In several synapses, Ca^{2+} signalling has been considered as one of the main actors in this arena! Almost every undergraduate student knows that elevating Ca^{2+} within neuronal cells is crucial to start the release of neurotransmitter! Indeed, Ca^{2+} is an ion that participates in almost everything within the steps of neurotransmitter release. However, its dysregulation may lead to toxic effects, growing into diseases like the neurological disorders. This concept is newer and, probably, not all students know it! Indeed, dysregulations of intracellular Ca^{2+} signalling achieved, for example, due to an excess of Ca^{2+} influx through voltage-activated Ca^{2+} channels, and yet disturbances of Ca^{2+} release from ryanodine and/or IP_3-sensitive intracellular Ca^{2+} stores have been reported in age-related animal models [2]. Several of these alterations in Ca^{2+} signalling, described in normal aging, can be replicated by exposing neurons to oxidative and metabolic stress in culture or *in vivo*, suggesting important contributions of essential aging mechanisms to the dysregulation of neuronal Ca^{2+} signalling in neurological disorders, such as in Alzheimer´s disease (AD). Furthermore, reports from brain tissue samples performed through brains of AD patients, and animal models of AD, have discovered significant changes in levels of proteins and genes directly related to neuronal Ca^{2+} signalling [2]. Not surprisingly, environmental factors that prevent amyloidogenesis (caloric restriction, cognitive stimulation, and antioxidants) alleviate neuronal Ca^{2+} signalling dysregulation, whereas factors that improve amyloidogenesis disrupt Ca^{2+} homeostasis.

As I stated in the beginning of this editorial, my current field of research involves the study of the interaction between Ca^{2+} and cAMP signalling pathways (Ca^{2+}/cAMP signalling interaction). Indeed, considering the dysregulation of Ca^{2+} signalling in neurological disorders, now became quite interesting the study of such interaction yet in neurological disorders. The cumulative knowledge in the field clearly accepts that ryanodine and/or IP_3-sensitive intracellular Ca^{2+} stores can be modulated by cAMP, whose rise within cells achieves the release of Ca^{2+} from these stores. As stated above, considering the excess of intracellular Ca^{2+} presented in neurological disorders, the levels of cAMP within neurons may also be dysregulated due to the Ca^{2+}/cAMP signalling interaction [3-10], thus yet affecting the release of Ca^{2+} from intracellular stores. Modern methodologies, which include fluorescence probes targeting Ca^{2+} and cAMP may provide novel insights in this arena! I am looking forward to obtaining these results!

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